Case report

Balloon dilatation of a stenosed bioprosthesis in the tricuspid valve position

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SUMMARY Percutaneous balloon dilatation of a stenosed Ionescu-Shiley bioprosthesis in the tricuspid valve position was performed twice in a 19 year old woman. On each occasion there was a considerable improvement in symptoms and haemodynamic function but the effect lasted for only a few months. When the valve was excised it showed no evidence of the previous balloon dilatations.

Case report

A 19 year old woman presented with a four month history of peripheral oedema, abdominal distension, breathlessness on exertion, and increasing lethargy. Her symptoms had progressed despite diuretic treatment. Soon after birth she had been found to have a ventricular septal defect with pulmonary valve stenosis. When she was six infective endocarditis developed on the tricuspid valve and this produced severe regurgitation. At operation the tricuspid valve was replaced with a 19 mm Björk-Shiley prosthesis, the ventricular septal defect was closed with a patch, and the pulmonary stenosis was relieved with an outflow patch. Four years later she had a further episode of endocarditis caused by Staphylococcus aureus. This was successfully treated medically. When she was 13 right heart failure developed. This was caused by subacute obstruction of the Björk-Shiley valve, which was replaced with a 29 mm Ionescu-Shiley bioprosthesis. Postoperatively bradycardia developed and because of this a permanent epicardial pacemaker was implanted. Atrial fibrillation developed when she was 18.

Physical examination showed an irregular pulse of 75 beats/min, a blood pressure of 115/70 mm Hg, bilateral pitting oedema to the waist, 15 cm elevation of the jugular venous pulse, 3 cm hepatomegaly, and mild ascites. A left parasternal heave was noted, and

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on auscultation both heart sounds were single, with a grade 2/6 pulmonary ejection murmur, a grade 1/6 pulmonary early diastolic murmur, and a grade 2/6 tricuspid diastolic murmur. The chest x ray showed cardiomegaly with right atrial enlargement and normal lung fields. The electrocardiogram showed atrial fibrillation with paced ventricular rhythm and occasional ventricular extrasystoles. The echocardiogram showed good left ventricular function and mildly impaired right ventricular function; and colour Doppler scanning showed a narrow, turbulent, high velocity jet through the tricuspid valve with minimal tricuspid valve regurgitation. There was no evidence of right ventricular outflow obstruction. Doppler measurements are given in the table.

Cardiac catheterisation was performed under local anaesthetic without sedation. Haemodynamic data are given in the table. The interatrial and interventricular septa were intact. The leaflets of the tricuspid valve prosthesis did not show calcification on fluoroscopy. A right ventricular angiogram showed slight dilatation of the ventricle and slightly impaired systolic function with a trace of tricuspid valve regurgitation.

A catheter with a 20 mm diameter balloon (Mansfield Scientific, Mansfield, MA) was introduced via the femoral vein. The balloon was centred through the prosthesis and inflated to maximum diameter on four occasions for 15–25 seconds (figure). During balloon inflation there was no fall in systemic arterial pressure, no impairment of consciousness, and no cyanosis—despite the fact that the interatrial and

Table Doppler echocardiographic and haemodynamic data

	First dilatation			Second dilatation		
	Before	After	Follow up	Before	After	Follow up
Weight (kg)	61.7	57.6*	55.0	61.4	57.4*	59.8
Heart rate (beats/min)	70	70	74	70	70	70
Blood pressure (mm Hg)	120/54	117/52	115/55	110/48	120/60	125/60
Doppler:	/	/		,	/	/
TVIT (ms)	748	493*	646	686	472*	635
Cardiac catheterisation:						
RA (mean) (mm Hg)	15	14		17	16	_
RV (mm Hg)	30/4	36/8	_	30/7	34/12	
TVEDG (mm Hg)	8–10	4–6		9–11	4–8	_
CI (l/min/m²)	3.4	3.2	_	2.8	2.8	
TVÄ (cm²)	0.52	0.65	_	0.43	0.58	

^{*}Measured 24 hours after dilatation.
CI, cardiac index; RA, right atrial pressure; RV, right ventricular pressure; TVA, tricuspid valve area; TVEDG, tricuspid valve end diastolic gradient; TV½T, tricuspid valve pressure half time.

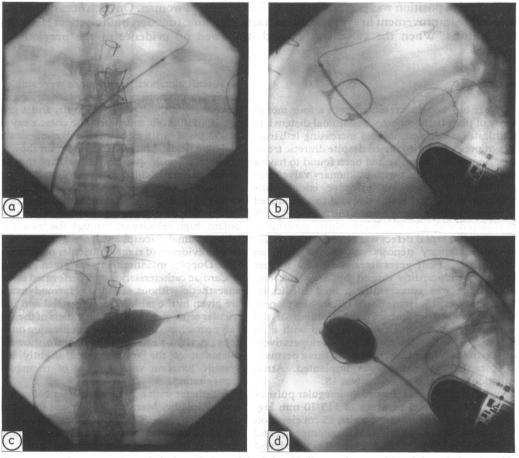


Figure (a) Anteroposterior and (b) lateral views of the deflated balloon catheter positioned through the tricuspid valve prosthesis. (c) During inflation the balloon reached a maximum diameter (20 mm) without "waisting" and (d) seemed to occlude the valve completely.

interventricular septa were intact and the balloon seemed to occlude the prosthetic valve completely. Haemodynamic measurements were repeated (table) and a repeat right ventricular angiogram showed no increase in the severity of the tricuspid regurgitation.

In the first 24 hours after the procedure the patient experienced a considerable diuresis and lost 4 kg in weight. At follow up five weeks later there had been a further reduction in weight with an appreciable improvement in symptoms. Over the following three months there was a gradual return of symptoms and an increase in weight to the first preadmission level. The physical signs of right heart failure with ascites had returned and Doppler echocardiography again confirmed severe stenosis of the tricuspid valve prosthesis (table).

The patient was readmitted and underwent repeat catheterisation. Haemodynamic data are given in the table. The balloon dilatation was repeated with a 23 mm diameter balloon, which was inflated to maximum diameter on two occasions for 30 seconds. Again there was no fall in systemic pressure during maximum balloon inflation. Repeat haemodynamic data are given in the table. A repeat right ventricular angiogram showed only a trace of tricuspid valve regurgitation.

After the second procedure there was a 4 kg weight loss overnight as a result of considerable diuresis. The improvement in symptoms was maintained for six months before the symptoms and signs of right heart failure returned. Further balloon dilatation was thought unlikely to produce a lasting improvement in symptoms and the patient underwent reoperation. The right atrium contained a large organised thrombus (7 cm \times 4 cm \times 4 cm). When the tricuspid prosthesis was excised its leaflets were found to be very stiff with calcific vegetations on both atrial and ventricular surfaces. There was no cusp fusion and no evidence of damage from balloon dilatation. A St Jude prosthesis was implanted and recovery was uneventful.

Discussion

Percutaneous balloon dilatation was offered to this patient as an alternative to valve replacement because she was young and had already undergone tricuspid valve replacement twice. The aim was to produce acceptable palliation rather than permanent relief of the valve stenosis. If the effect of balloon dilatation had persisted, insertion of a mechanical prosthesis

(necessitating oral anticoagulation) might have been delayed until after childbearing.

The mechanism of the partial relief of the obstruction is unclear. The leaflets of the bovine pericardial prosthesis were very stiff and balloon dilatation probably stretched or otherwise reduced the stiffness of the valve leaflets.

Balloon dilatation of a tricuspid valve prosthesis has been reported once before. Feit et al produced apparently successful palliation in a patient with a 31 mm Hancock porcine prosthesis. Their patient was followed up for only one month and so it is not clear whether the benefit persisted. They noted a fall in right atrial pressure and an increase in cardiac index, whereas in our patient there was no change in cardiac index and we saw an increase in right ventricular end diastolic pressure rather than a fall in right atrial pressure after balloon dilatation.

McKay et al reported two patients who underwent balloon dilatation of an aortic valve bioprosthesis.² The procedure was complicated by severe damage to the prosthesis and one patient died as a result. They suggest that contraindications to dilatation of prosthetic valves should include valve leaflet calcification, the presence of vegetations or thrombosis, and degenerative changes in the valve cusps.

Lloyd et al reduced valvar gradients in three of five patients undergoing balloon dilatation of calcified stenotic bioprosthetic valves in right ventricle to pulmonary artery conduits but no follow up data were given.³

Our experience suggests that the role of balloon dilatation in the management of stenosed bioprosthetic valves will be limited. Although symptoms and haemodynamic function may be improved, lasting benefit is unlikely.

References

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